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=> s galectin-3
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=> s galectin-3
L1 1767 GALECTIN-3

=> s l1 (p) inhibitor
L2 93 L1 (P) INHIBITOR

=> duplicate remove l2
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
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PROCESSING COMPLETED FOR L2
L3 33 DUPLICATE REMOVE L2 (60 DUPLICATES REMOVED)

=> s l3 (p) composition
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L22 (P) COMPOSITI'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L26 (P) COMPOSITI'
L4 0 L3 (P) COMPOSITION

=> d l3 1-33 ibib abs

L3 ANSWER 1 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:276136 CAPLUS
DOCUMENT NUMBER: 136:273164
TITLE: Drug screening for inhibitors of Ras binding to galectins and their therapeutic use as anti-tumor agents
INVENTOR(S): Kloog, Yoel; Haklai, Roni; Paz, Ariella; El Ad-Sfadia, Galit; Ballan, Eyal
PATENT ASSIGNEE(S): Ramot University Authority for Applied Research & Industrial Development Ltd., Israel
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002029031	A2	20020411	WO 2001-IL918	20011001

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-237858P P 20001004

AB Disclosed is a method for identifying cell membrane anchor proteins that bind Ras protein. Also disclosed are methods for identifying other anchor proteins that bind isoforms of Ras, methods of identifying drug candidates that inhibit aberrant Ras activity and methods of detg. therapeutic dosages of the drugs. These methods involve crosslinking cell membrane exts. contg. Ras proteins both in the presence or absence of Ras antagonists and identifying crosslinked complexes in the absence of Ras antagonists. Ras genes, frequently mutated in human tumors, promote malignant transformation. Ras transformation requires membrane anchorage, which is promoted by Ras farnesyl-cysteine carboxymethylester and by a second signal. The Ras antagonist may be an ***inhibitor*** of a prenylated, non-prenylated or a farnesylated Ras protein such as S-trans, trans-farnesylthiosalicylic acid (FTS) or an analog. Cell membranes may be fibroblasts transformed with oncogenic K-Ras, H-Ras or N-Ras, 518A2/N-Ras melanoma cells, 607B melanoma cells, Panc-1 cells contg. oncogenic K-Ras, EJ cells contg. H-Ras or MC-MA-11 cells. The crosslinking agents may be DSS or DSP. Ras activity is detd. by measuring the effect of the said antagonist on the dimerization of Ras, change in the activation of Raf or in the binding of Raf to Ras or by measuring the change in binding between the Ras protein and the anchor protein. Furthmore, the Ras protein or the anchor protein may be immobilized on a matrix and labeled with a fluorescent protein like green or yellow fluorescent protein. Anchor proteins may be galectin-1, ***galectin*** - ***3*** , galectin-7 or galectin-8. The loss of binding between Ras protein and anchor protein may also be measured which comprises measuring the intracellular movement of the Ras protein or the anchor protein. Aberrant Ras activity may be inhibited by treating a patient with an antisense oligonucleotide which prevents expression of a gene encoding for a Ras anchor protein. Such an oligonucleotide may contain a phosphorathioate-modified nucleotide and the said antisense oligonucleotide may be administered to a patient via a liposome. A method for detg. the effective dosage of a Ras antagonist is also provided by measuring the decrease in anchor protein concn., following treatment of cell membrane ext. with a Ras antagonist.

L3 ANSWER 2 OF 33 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2002142265 MEDLINE

DOCUMENT NUMBER: 21850619 PubMed ID: 11724777

TITLE: Galectin-3 phosphorylation is required for its anti-apoptotic function and cell cycle arrest.

AUTHOR: Yoshii Tadashi; Fukumori Tomoharu; Honjo Yuichiro; Inohara Hidenori; Kim Hyeong-Reh Choi; Raz Avraham

CORPORATE SOURCE: Tumor Progression and Metastasis Program, Karmanos Cancer Institute, Wayne State University, 110 E. Warren Ave., Detroit, MI 48201, USA.

CONTRACT NUMBER: CA 46120 (NCI)

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Mar 1) 277 (9) 6852-7.
Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200204

ENTRY DATE: Entered STN: 20020307
Last Updated on STN: 20020403
Entered Medline: 20020401

AB ***Galectin*** - ***3*** , a beta-galactoside-binding protein, is

implicated in cell growth, adhesion, differentiation, and tumor progression by interactions with its ligands. Recent studies have revealed that ***galectin*** - ***3*** suppresses apoptosis and anoikis that contribute to cell survival during metastatic cascades. Previously, it has been shown that human ***galectin*** - ***3*** undergoes post-translational signaling modification of Ser(6) phosphorylation that acts as an "on/off" switch for its sugar-binding capability. We questioned whether ***galectin*** - ***3*** phosphorylation is required for its anti-apoptotic function. Serine to alanine (S6A) and serine to glutamic acid (S6E) mutations were produced at the casein kinase I phosphorylation site in ***galectin*** - ***3*** . The cDNAs were transfected into a breast carcinoma cell line BT-549 that innately expresses no ***galectin*** - ***3*** . Metabolic labeling revealed that only wild type ***galectin*** - ***3*** undergoes phosphorylation in vivo. Expression of Ser(6) mutants of ***galectin*** - ***3*** failed to protect cells from cisplatin-induced cell death and poly(ADP-ribose) polymerase from degradation when compared with wild type ***galectin*** - ***3*** . The non-phosphorylated ***galectin*** - ***3*** mutants failed to protect cells from anoikis with G(1) arrest when cells were cultured in suspension. In response to a loss of cell-substrate interactions, only cells expressing wild type ***galectin*** - ***3*** down-regulated cyclin A expression and up-regulated cyclin D(1) and cyclin-dependent kinase ***inhibitors*** , i.e. p21(WAF1/CIP1) and p27(KIP1) expression levels. These results demonstrate that ***galectin*** - ***3*** phosphorylation regulates its anti-apoptotic signaling activity.

L3 ANSWER 3 OF 33 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2002342426 IN-PROCESS
DOCUMENT NUMBER: 22080296 PubMed ID: 12083851
TITLE: Role of elastin-matrix interactions in tumor progression.
AUTHOR: Lapis Karoly; Timar Jozsef
CORPORATE SOURCE: First Department of Pathology and Experimental Cancer Research, Semmelweis University, Budapest, H-1085, Hungary.
SOURCE: SEMINARS IN CANCER BIOLOGY, (2002 Jun) 12 (3) 209-17. Journal code: 9010218. ISSN: 1044-579X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020627
Last Updated on STN: 20020627
AB Data from the literature now indicate that cancer cells can specifically interact with the unique extracellular matrix protein, elastin. The interaction is mediated by two elastin-binding proteins (EBP), S-gal/EBP (organized into the elasin receptor/elastonectin complex) and ***galectin*** - ***3*** , components of two laminin receptors. Studies revealed that the expression of both EBPs is closely associated to the invasive/metastatic potential of various cancer types. This is due to the fact that elastin-ligation of S-gal/EBP induces motogenic, as well as mitogenic signals and releases various elastases from cancer cells and the induction depends on the metastatic potential. Studies also demonstrated that certain cancer cells can synthesize elastin and express lysyl oxydase, providing explanation for frequent appearance of elastic tissue in tumors such as breast or gastric cancers. Clinico-pathological data suggest some correlation with tumor progression of the presence of the elastic tumor stroma. Since elastic tissue may be a significant reservoir of angiostatic molecule(s) this extracellular matrix protein can also have a role in tumor-induced angiogenesis. Soluble elastin as well as elastin peptides are potent ***inhibitors*** of the metastatic process in experimental tumor models. On the other hand, elastin peptides can also be used to design targeted therapies exploiting the unique physicochemical nature of this matrix protein. Altogether, these data suggest a significant role for tumor cell-elastin interactions in tumor progression.

L3 ANSWER 4 OF 33 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2002192081 MEDLINE
DOCUMENT NUMBER: 21916672 PubMed ID: 11921396
TITLE: Low micromolar ***inhibitors*** of ***galectin*** - ***3*** based on 3'-derivatization of N-acetyllactosamine.
AUTHOR: Sorme Pernilla; Qian Yuning; Nyholm Per-Georg; Leffler

Hakon; Nilsson Ulf J
CORPORATE SOURCE: Section MIG (Microbiology, Immunology, Glycobiology), Dept. of Laboratory Medicine, Lund University, Solvegatan 23, 22362 Lund, Sweden.
SOURCE: Chembiochem, (2002 Mar 1) 3 (2-3) 183-9.
Journal code: 100937360. ISSN: 1439-4227.
PUB. COUNTRY: Germany: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020403
Last Updated on STN: 20020619
Entered Medline: 20020618

AB A strategy for generating potential galectin ***inhibitors*** was devised based on derivatization at the C-3' atom in 3'-amino-N-acetyllactosamine by using structural knowledge of the galectin carbohydrate recognition site. A collection of 12 compounds was prepared by N-acylations or N-sulfonylations. Hydrophobic tagging of the O-3 atom in the N-acetylglucosamine residue with a stearic ester allowed rapid and simple product purification. The compounds were screened in a ***galectin*** - ***3*** binding assay and three compounds with significantly higher inhibitory activities compared to the parent N-acetyllactosaminide were found. These three best ***inhibitors*** all carried an aromatic amide at the C-3' position of the galactose moiety, which indicates that favorable interactions were formed between the aromatic group and ***galectin*** - ***3*** . The best ***inhibitor*** had an IC50 value (4.4 microM) about 50 times better than the parent N-acetyllactosaminide, which implies that it has potential as a valuable tool for studying ***galectin*** - ***3*** biological functions and also as a lead compound for the development of ***galectin*** - ***3*** -blocking pharmaceuticals.

L3 ANSWER 5 OF 33 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2002154636 IN-PROCESS
DOCUMENT NUMBER: 21883828 PubMed ID: 11886846
TITLE: Galectin-3 is strongly up-regulated in nonapoptosing mammary epithelial cells during rat mammary gland involution.
AUTHOR: Mengwasser Jorg; Liu Fu-Tong; Sleeman Jonathan P
CORPORATE SOURCE: Forschungszentrum Karlsruhe, Institute for Toxicology and Genetics, PO Box 3640, D-76021 Karlsruhe, Germany, and Division of Allergy, La Jolla Institute for Allergy and Immunology, 10355 Science Center Drive, San Diego, CA 92121, USA.
SOURCE: GLYCOBIOLOGY, (2002 Feb) 12 (2) 129-34.
Journal code: 9104124. ISSN: 0959-6658.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020312
Last Updated on STN: 20020312

AB ***Galectin*** - ***3*** is an endogenous mammalian lectin that binds to ABH carbohydrate antigens. Here we show that ***galectin*** - ***3*** is strongly up-regulated during mammary gland involution and that it is expressed virtually exclusively on nonapoptotic cells. We demonstrate that dexamethasone, an ***inhibitor*** of the second phase of mammary gland involution, potently suppresses up-regulation of ***galectin*** - ***3*** as judged immunohistochemically and on western blots, suggesting that systemic hormone levels regulate ***galectin*** - ***3*** expression during involution. However, at the RNA level ***galectin*** - ***3*** expression is rapidly up-regulated on the onset of involution but remains consistantly high during the first and second phase of involution regardless of dexamethasone treatment. These data suggest that the up-regulation of ***galectin*** - ***3*** in the involuting mammary gland is not only controlled transcriptionally but also regulated posttranscriptionally under the control of systemic glucocorticoid hormones involved in coordinating the involution process.

L3 ANSWER 6 OF 33 CAPLUS

ACCESSION NUMBER: 2002:179383 CAPLUS
TITLE: N.epsilon.-(carboxymethyl)lysine-induced angial cell activation
AUTHOR(S): Lim, Hyun Jin; Song, Jaesook; Ha, Hunjoo; Lee, Hi Bahl
CORPORATE SOURCE: Department of Internal Medicine, Hyonam Kidney Laboratory, College of Medicine, Soon Chun Hyang University, Seoul, S. Korea
SOURCE: Taehan Sinjang Hakhoechi (2002), 21(1), 20-28
CODEN: TSHACY; ISSN: 1225-0015
PUBLISHER: Korean Society of Nephrology
DOCUMENT TYPE: Journal
LANGUAGE: Korean

AB Background:Advanced glycation end products (AGE) are independent risk factors in the development and progression of diabetic nephropathy. Receptor for AGE(RAGE) is considered the main receptor involved in AGE-induced cell activation. ***Galectin*** - ***3*** , another AGE receptor, has recently been found up-regulated in mesangial cells(MC) cultured under high glucose and in diabetic rat kidneys. N.epsilon.-(carboxymethyl)lysine(CML) is a well characterized AGE but its role in MC activation is unknown. The present study examd. the effects of CML on MC proliferation and extracellular matrix(ECM) secretion. Methods: Synchronized rat MC were stimulated with different concns. of CML-bovine serum albumin(BSA), control BSA, and transforming growth factor-.beta.1(TGF-.beta.1) for up to 72 h. Cell proliferation was measured by [3H]-thymidine incorporation. Fibronectin, TGF-.beta.1, plasminogen activator ***inhibitor*** (PAI)-1 secreted into the media and RAGE and ***galectin*** - ***3*** expression in MC were measured by Western blot anal. and ELISA Results: 1,000 .mu.g/mL of CML-BSA decreased [3H]-thymidine incorporation by MC at 48 h and 10 ng/mL TGF-.beta.1 at 24 and 48 h. CML-BSA 100 and 1,000 pg/mL, control BSA 1,000 pg/mL, and TGF 8 10 ng/mL increased fibronectin secretion at 48 h CML-BSA up to 1,000 pg/mL did not affect TGF B1 or PAI-1 secretion. TGF-.beta.1 10 ng/mL, however, significantly increased PAI-1 secretion. Cultured MC expressed both RAGE and galec- tin-3. CML-BSA 100 .mu.g/mL upregulated ***galectin*** - ***3*** expression. Conclusion: CML-BSA decreased MC proliferation and increased fibronectin secretion, suggesting that CML may lead to ECM accumulation and glomerulosclerosis in diabetic animals. MC express RAGE and ***galectin*** - ***3*** constitutively and CML-induced ***galectin*** - ***3*** upregulation may have a role in AGE-induced MC activation.

L3 ANSWER 7 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:397050 CAPLUS
DOCUMENT NUMBER: 135:1241
TITLE: Regulated expression constructs for cyclin dependent kinase inhibitor genes and their use in identification of genes regulated by them
INVENTOR(S): Chang, Bey-dih; Roninson, Igor B.
PATENT ASSIGNEE(S): Board of Trustees of the University of Illinois, USA
SOURCE: PCT Int. Appl., 136 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001038532	A2	20010531	WO 2000-US28082	20001011
WO 2001038532	A3	20011227		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO 2000061751	A1	20001019	WO 2000-US9286	20000407

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,

JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 1999-449589 A2 19991129
WO 2000-US9286 A2 20000407
US 1999-128676P P 19990409

AB This invention provides methods and reagents for identifying genes involved in cell cycle progression, growth promotion, modulation of apoptosis, cellular senescence and aging, and methods for identifying compds. that inhibit or potentiate cellular senescence. Specifically, genes for p16 or p21 cyclin-dependent kinase inhibitors (CDK inhibitors) are placed under the control of promoters with known patterns of regulation and the effects of expression of genes for the CDK inhibitors on patterns of gene expression and cellular phenotypes are detd. These expression constructs can be used to screen for effectors of the inhibitors that can be used to control the cell cycle and cell aging or apoptosis. HT-1080 cells were transformed with an expression construct for a p21 from a cytomegalovirus promoter under control of the lac repressor. This allowed lactose-dependent expression of the p21 gene. Induction of the gene led to a loss of clonogenicity and to an increased no. of abnormal mitotic figures and endoreduplication. A no. of genes that were induced or repressed by p21 expression were identified and patterns of regulation by other stimuli were studied..

L3 ANSWER 8 OF 33 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2001342993 MEDLINE
DOCUMENT NUMBER: 21299139 PubMed ID: 11406562
TITLE: The role of Thomsen-Friedenreich antigen in adhesion of human breast and prostate cancer cells to the endothelium.
AUTHOR: Glinsky V V; Glinsky G V; Rittenhouse-Olson K; Huflejt M E; Glinskii O V; Deutscher S L; Quinn T P
CORPORATE SOURCE: Department of Biochemistry, University of Missouri, Columbia, Missouri 65211, USA.
SOURCE: CANCER RESEARCH, (2001 Jun 15) 61 (12) 4851-7.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010716
Last Updated on STN: 20010716
Entered Medline: 20010712

AB Interactions of metastatic cancer cells with vasculatory endothelium are critical during early stages of cancer metastasis. Understanding the molecular underpinnings of these interactions is essential for the development of new efficacious cancer therapies. Here we demonstrate that cancer-associated carbohydrate T antigen plays a leading role in docking breast and prostate cancer cells onto endothelium by specifically interacting with endothelium-expressed beta-galactoside-binding protein, ***galectin*** - ***3*** . Importantly, T antigen-bearing glycoproteins are also capable of mobilizing ***galectin*** - ***3*** to the surface of endothelial cells, thus priming them for harboring metastatic cancer cells. The T antigen-mediated, tumor-endothelial cell interactions could be efficiently disrupted using synthetic compounds either mimicking or masking this carbohydrate structure. High efficiency of T antigen-mimicking and T antigen-masking ***inhibitors*** of tumor cell adhesion warrants their further development into antiadhesive cancer therapeutics.

L3 ANSWER 9 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:79677 CAPLUS
DOCUMENT NUMBER: 134:234948
TITLE: Simultaneous Induction of Galectin-3 Phosphorylated on Tyrosine Residue, p21WAF1/Cip1/Sdi1, and the Proliferating Cell Nuclear Antigen at a Distinctive Period of Repair of Hepatocytes Injured by CCl4
AUTHOR(S): Yamazaki, Kazumaro; Kawai, Akiko; Kawaguchi, Makoto;

Hibino, Yasuhide; Li, Fang; Sasahara, Masakiyo; Tsukada, Kazuhiro; Hiraga, Koichi

CORPORATE SOURCE: Department of Biochemistry, Toyama Medical and Pharmaceutical University School of Medicine, Toyama, 930-0194, Japan

SOURCE: Biochemical and Biophysical Research Communications (2001), 280(4), 1077-1084
CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The period of repair of hepatocytes injured by CCl4 and signaling proteins intrinsic to this period were examd. A 30 kDa polypeptide detected by immunoblot anal. using anti-phosphotyrosine antibody in livers from rats 48 to 72 h after administration of a single dose of CCl4 was identified as galectin-3 induced in cytoplasm of periportal hepatocytes and phosphorylated on tyrosine residue(s). Simultaneously, these hepatocytes induced p21WAF1/Cip1/Sdi1 in the nucleus and the proliferating cell nuclear antigen in both the nucleus and the cytoplasm, suggesting that hepatocytes during this distinctive period are quiescent and repair cellular damage. Trabecular architecture of hepatocytes with the proliferating cell nuclear antigen only in the nucleus was found at 96 h. These findings indicate that galectin-3 is a novel member of signaling proteins downstream of tyrosine kinase, and suggest that it plays roles in supporting repair or survival of the injured hepatocytes rather than their proliferation that is likely to be initiated later than 72 h. (c) 2001 Academic Press.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 33 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:244306 BIOSIS

DOCUMENT NUMBER: PREV200100244306

TITLE: Identification of a glycosylated isoform of Bcl-2.

AUTHOR(S): Chaney, William G. (1); Fernandes-Paul, Mirabella E. (1)

CORPORATE SOURCE: (1) Biochemistry and Molecular Biology, U. Nebraska Med. Ctr., 984525 Nebraska Medical Center, Omaha, NE, 68198-4525 USA

SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A870. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Galectin 3 has been reported to bind to Bcl-2 in Jurkat cell extracts (Yang et al, Proc. Nat. Acad. Sci, USA, 93:6737). The present study confirms this report for Jurkat- and MCF7-derived Bcl-2 and establishes that VVA isolectin B4, the N-Acetylgalactosamine binding lectin from Vicia villosa, can also bind to Bcl-2. Lactose, but not sucrose inhibits Bcl-2 binding to both lectins. Treatment of cell extracts with periodate or with N-Acetylgalactosaminidase destroys the ability of Bcl-2 to bind to either galectin 3 or VVA. Bcl-2 expressed in E. coli failed to bind to either lectin, demonstrating that Bcl-2 protein alone is not sufficient for binding. Sequential precipitation of extracts showed that approximately 5% of cellular Bcl-2 could be bound by VVA and that the galectin 3 and VVA interact with the same Bcl-2 isoform. These studies demonstrate the presence of a post-translationally glycosylated isoform of Bcl-2 bearing a terminal N-Acetylgalactosamine residue that is an endogenous cellular ligand of galectin 3.

L3 ANSWER 11 OF 33 MEDLINE DUPLICATE 6

ACCESSION NUMBER: 2002210350 MEDLINE

DOCUMENT NUMBER: 21942287 PubMed ID: 11948868

TITLE: Wedgelike glycodendrimers as inhibitors of binding of mammalian galectins to glycoproteins, lactose maxiclusters, and cell surface glycoconjugates.

AUTHOR: Andre S; Pieters R J; Vrasidas I; Kaltner H; Kuwabara I; Liu F T; Liskamp R M; Gabius H J

CORPORATE SOURCE: Institut fur Physiologische Chemie, Tierarztliche Fakultat,

Ludwig-Maximilians-Universitat Munchen, Veterinarstrasse 13, 80539 Munchen, Germany.
SOURCE: Chembiochem, (2001 Nov 5) 2 (11) 822-30.
Journal code: 100937360. ISSN: 1439-4227.
PUB. COUNTRY: Germany: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020412
Last Updated on STN: 20020502
Entered Medline: 20020501

AB Galectins are mammalian carbohydrate-binding proteins that are involved in cell-cell and cell-matrix adhesion, cell migration, and growth regulation with relevance to inflammation and tumor spread. These important functions account for the interest to design suitable low molecular weight ***inhibitors*** that match the distinct modes of presentation of the carbohydrate recognition domains of the different galectin subfamilies. Using 3,5-di-(2-aminoethoxy)benzoic acid as the branching unit, wedgelike glycodendrimers with two, four, and eight lactose moieties (G1-G3) were synthesized. They were tested in solid-phase competition assays with lactose maxiclusters and various N-glycan branching profiles (miniclusters) as the matrix and also in cell assays. Prototype galectins-1 and -7, chimera-type ***galectin*** - ***3*** , a plant (AB)(2) toxin, and a lactose-binding immunoglobulin G fraction from human serum were the carbohydrate-binding targets. Potent inhibition and remarkable cluster effects were seen for the homodimeric galectin-1, especially in combination with biantennary N-glycans as the matrix. Remarkably, for the tetravalent G2 glycodendrimer, the inhibitory potency of each lactose unit reached a maximum value of 1667 relative to free lactose. In haemagglutination experiments as a model for cell adhesion, ***galectin*** - ***3*** was markedly sensitive to increased sugar valency and a relative potency per lactose of 150 was reached. The spatial orientation of the carbohydrate recognition domains of the endogenous lectins and the branching pattern of the carbohydrates of the glycoprotein matrices used are both important factors in the design and synthesis of glycodendrimers with galectin-selective properties.

L3 ANSWER 12 OF 33 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:576840 SCISEARCH
THE GENUINE ARTICLE: 453XR
TITLE: Identification of differentially expressed genes in hepatocellular carcinoma and metastatic liver tumors by oligonucleotide expression profiling
AUTHOR: Tackels-Horne D; Goodman M D; Williams A J; Wilson D J; Eskandari T; Vogt L M; Boland J F; Scherf U; Vockley J G (Reprint)
CORPORATE SOURCE: Gene Log Inc, 708 Quince Orchard Rd, Gaithersburg, MD 20878 USA (Reprint); Gene Log Inc, Gaithersburg, MD 20878 USA
COUNTRY OF AUTHOR: USA
SOURCE: CANCER, (15 JUL 2001) Vol. 92, No. 2, pp. 395-405.
Publisher: JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA.
ISSN: 0008-543X.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 39

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB BACKGROUND. The characterization of differentially expressed genes between cancerous and normal tissues is an important step in the understanding of tumorigenesis. Global gene expression profiling with microarrays has now offered a powerful tool to measure the changes of thousands of genes in any carcinoma tissues in an effort to identify these key disease-related genes. To compare the gene expression of a primary liver carcinoma, metastatic carcinoma to the liver, and normal liver, the authors analyzed tissue from six primary hepatocellular carcinomas (HCCs), five colorectal adenocarcinoma metastases to the liver, and eight normal livers.

METHODS. Samples were processed from total RNA to fragmented cRNA and hybridized onto Affymetrix GeneChip((R)) expression arrays. Analyses were performed to determine the consensus pattern of gene expression for

primary liver carcinoma, metastatic liver carcinoma, and normal liver tissue and their changes in expression level.

RESULTS. In hepatocellular carcinoma, 842 genes were overexpressed, and 393 genes were underexpressed in comparison with genes of normal liver tissue. Of note, 7 of the 20 most increased identified known genes previously have been associated with liver carcinoma or other types of cancers. The 13 additional identified genes until now have not previously shown strong association with cancers. Furthermore, the authors identified 42 genes and 24 expressed sequence tags that are expressed at a significant level in both HCC and metastastic tumors, presenting a list of marker genes indicative of cancerous liver tissue.

CONCLUSIONS. In this study, genes that can be involved in the production of and maintenance of hepatic carcinomas were identified. These data offer new insight into genes that are potentially important in the pathogenesis of liver carcinoma, as well as additional targets for new strategies for cancer therapy and treatment. (C) 2001 American Cancer Society.

L3 ANSWER 13 OF 33 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2002031556 MEDLINE
DOCUMENT NUMBER: 21595829 PubMed ID: 11759230
TITLE: [Rheumatoid arthritis: new developments in the pathogenesis with special reference to synovial fibroblasts].
Die Rheumatoide Arthritis: Neuentwicklungen in der Pathogenese unter besonderer Berucksichtigung der synovialen Fibroblasten.
AUTHOR: Seemayer C A; Distler O; Kuchen S; Muller-Ladner U; Michel B A; Neidhart M; Gay R E; Gay S
CORPORATE SOURCE: WHO-Collaborating Center for Molecular Biology and Novel Therapeutic Strategies of Rheumatic Diseases, Department of Rheumatology, University Hospital Zurich, Gloriastrasse 25, 8091 Zurich, Switzerland.
SOURCE: ZEITSCHRIFT FUR RHEUMATOLOGIE, (2001 Oct) 60 (5) 309-18. Ref: 67
Journal code: 0414162. ISSN: 0340-1855.
PUB. COUNTRY: Germany: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020124
Last Updated on STN: 20020131
Entered Medline: 20020130

AB Rheumatoid arthritis (RA) is a chronic inflammatory disease, which is mainly characterized by synovial hyperplasia, pathological immune phenomena and progressive destruction of the affected joints. Various cell types are involved in the pathogenesis of RA including T cells, antigen presenting cells, and endothelial cells. Recent experimental evidence suggests that the CD40/CD154 system might play an important role in the development of RA. Our experimental approach focuses on RA synovial fibroblasts (RA-SF) that are able to destroy articular cartilage independent of inflammation. To elucidate the specific role of those cells in RA pathophysiology the following questions are currently addressed: 1. Which mechanisms do activate the RA-SF? 2. How do the activated RA-SF attach to the cartilage? 3. How do RA-SF destroy cartilage and bone? Which mechanisms do activate the RA-SF? The process of activation is poorly understood. It is unclear, how far the synovial hyperplasia of RA resembles tumor diseases. Along this line some contradictory results exist concerning the role of the tumor suppressor protein p53. Some investigations could show the expression of p53 in the synovial lining including p53 mutations in RA synovium and in RASF, while other research groups could not confirm these data. Our group has demonstrated that the tumor suppressor PTEN was less expressed in the synovial lining of RA than in normal synovium, but no PTEN mutations could be found in the RA-SF. In addition, the in vivo and in vitro expression of the anti-apoptotic molecule sentrin suggests a functional resistance of RA-SF to undergo apoptosis. Although it is still unclear, whether certain viruses or viral elements are involved in the pathogenesis of RA (cause, consequence or coincidence?), certain viruses could play a role in the pathogenesis of RA. The endogenous retroviral element L1 was found to be expressed in the

synovial lining, at sites of invasion as well as in RA-SF grown in vitro. Moreover, the data indicate that after the initial activation of L1 downstream molecules such as the SAP kinase 4, the met-protooncogene and the ***galectin*** - ***3*** binding protein are upregulated. How do the activated RA-SF attach to the cartilage? It has been suggested that integrins mediate the attachment of RA-SF to fibronectin rich sites of cartilage. Intriguingly, other adhesion molecules such as the vascular cellular adhesion molecule-1 (VCAM) and CS-1, a splice variant of fibronectin, are synthesized by RA-SF. By binding to these adhesion molecules, lymphocytes that express the integrin VLA-4 could be stimulated and thereby maintain the inflammatory process. Osteopontin is an extracellular matrix protein, which is associated with matrix adhesion and metastasis in tumors. In RA synovium, osteopontin was detectable in the synovial lining and at sites of invasion. How do RA-SF destroy cartilage and bone? The destruction of cartilage and bone in RA is mediated by matrix metalloproteinases (MMPs) and cathepsins. MMPs exist as secreted and as membrane bound forms. In vitro models are being developed to simulate the invasive process of RA-SF. In an in vitro model developed in our laboratory, the treatment of RA-SF with anti-CD44 or anti-interleukin-1 (IL-1) minimized matrix degradation of RA-SF. On the other hand, co-culture of RA-SF and U937 cells as well as application of interleukin-1 beta (IL-1 beta) or tumor necrosis factor alpha (TNF alpha) increased the invasiveness of RA-SF. Gene transfer of bovine pancreas trypsin ***inhibitor*** (BPMI) or interleukin-10 (IL-10) reduced the invasion of RA-SF, while transduction of interleukin-1 receptor antagonist (IL-1Ra) was chondroprotective. Double gene transfer of IL-10 and IL-1Ra resulted in both inhibition of invasion and chondroprotection.

L3 ANSWER 14 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:562715 CAPLUS
DOCUMENT NUMBER: 136:230881
TITLE: An antibody to p16INK4A recognizes a modified form of galectin-3
AUTHOR(S): Gump, Jay; Koh, James
CORPORATE SOURCE: Department of Molecular Physiology and Biophysics, University of Vermont, Burlington, VT, USA
SOURCE: Hybridoma (2001), 20(3), 167-174
CODEN: HYBRDY; ISSN: 0272-457X
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Galectin-3 is a carbohydrate binding protein involved in multiple processes including cell-cycle regulation and apoptosis. The ability of galectin-3 to protect cells from apoptosis is dependent upon a region of the protein known as a BH-1 domain for its homol. to the anti-apoptotic protein Bcl-2. Here, we show that a monoclonal antibody (MAb) to the human tumor suppressor protein p161NK4A recognizes a post-translationally modified form of human galectin-3. The modified form is detectable in only a subset of cell types expressing galectin-3, indicating that the modification is cell-type-specific. Although there is little amino acid sequence homol. between p161NK4a and galectin-3, we show by epitope mapping that the modification directly affects the structure of galectin-3's BH-1 domain. Elucidation of the nature of this modification might provide further insight into galectin-3 function.
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:742378 CAPLUS
DOCUMENT NUMBER: 133:307317
TITLE: Galectin-3 expression is induced in cirrhotic liver and hepatocellular carcinoma
INVENTOR(S): Hsu, Daniel K.; Liu, Fu-Tong; Dowling, Christopher A.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2000062076	A1	2000[illegible]19	WO 2000-US8561	2000[illegible]9	

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-129111P P 19990413

AB The present invention relates to the discovery of a marker for liver disease. Novel diagnostics, prognostics, therapeutics and methods of use of the foregoing for the treatment and prevention of hepatocellular carcinoma are also disclosed. The expression of galectin-3 in normal human liver and cirrhotic human liver biopsy tissue was analyzed by immunohistochem. staining.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 16 OF 33 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:742246 CAPLUS

DOCUMENT NUMBER: 133:291954

TITLE: An inducible expression vector for the p21 cyclin-dependent kinase inhibitor and its use in identifying genes regulated by p21

INVENTOR(S): Chang, Bey-Dih; Roninson, Igor B.

PATENT ASSIGNEE(S): Board of Trustees of the University of Illinois, USA

SOURCE: PCT Int. Appl., 119 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000061751	A1	20001019	WO 2000-US9286	20000407

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1169443	A1	20020109	EP 2000-920212	20000407

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

WO 2001038532	A2	20010531	WO 2000-US28082	20001011
WO 2001038532	A3	20011227		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-128676P P 19990409
US 1999-449589 A2 19991129
WO 2000-US9286 W 20000407

AB This invention provides methods and reagents for identifying genes involved in cell cycle progression, growth promotion, modulation of apoptosis, cellular senescence and aging, and methods for identifying compds. that inhibit or potentiate cellular senescence, regulated by p21. The method uses an expression vector in which the p21CIP1/WAF1 gene is placed under control of an inducible promoter. The regulated expression

construct can be used to study the effects of p21 on the expression of a reporter gene from a given promoter. The reporter gene can also be used to screen for effectors of p21 function.
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 17 OF 33 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 2001079845 MEDLINE
DOCUMENT NUMBER: 21017431 PubMed ID: 11145021
TITLE: Retrotransposable L1 elements expressed in rheumatoid arthritis synovial tissue: association with genomic DNA hypomethylation and influence on gene expression.
AUTHOR: Neidhart M; Rethage J; Kuchen S; Kunzler P; Crowl R M; Billingham M E; Gay R E; Gay S
CORPORATE SOURCE: Center for Experimental Rheumatology, Department of Rheumatology, University Hospital, Zurich, Switzerland.
SOURCE: ARTHRITIS AND RHEUMATISM, (2000 Dec) 43 (12) 2634-47. Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010111
AB OBJECTIVE: Rheumatoid arthritis (RA) is characterized by a progressive destruction of joints by invasive synovial fibroblasts (SF). We searched for retroviral sequences in RA synovial fluid pellets, identified a sequence similar to that of open reading frame 2 (ORF2)/L1 retrotransposable elements, explored the expression of L1 in RA synovial tissues and cultured RA SF, and investigated the link to genomic DNA hypomethylation and the influence of functional L1 on gene expression. METHODS: RA synovial fluid pellets were screened by reverse transcriptase-polymerase chain reaction (RT-PCR) using degenerated pol primers. The sequences were identified by GenBank search. Riboprobes to ORF2/L1 and ***galectin*** - ***3*** and antibodies to the ORF1/L1-related p40 protein were used for in situ hybridization and immunohistochemistry of synovial tissues and cultured RA SF. Real-time quantitative RT-PCR was used for detecting ORF1 messenger RNA (mRNA). Since DNA hypomethylation occurs in inflammatory diseases, we incubated cells with the methylation ***inhibitor*** 5-aza-2'-deoxycytidine (5-azaC) and compared RA SF and osteoarthritis (OA) SF. L1-negative RA SF were transfected with the functional L1.2 construct, and differential gene expression was analyzed by subtractive hybridization combined with nested PCR. RESULTS: RNA sequences similar to those of ORF2/L1 retrotransposable elements, THE1 transposon, human endogenous retrovirus (ERV)-E, human ERV-HC2, and gibbon ape leukemia virus pol genes were isolated from different RA synovial fluid pellets. In RA synovial tissues, ORF2/L1 transcripts were detected in the sublining layer and at sites of cartilage and bone destruction. ***Galectin*** - ***3*** mRNA and L1-related ORF1/ p40 protein showed similar expression patterns. In contrast, OA synovial tissues in situ and cultures in vitro were negative. Real-time quantitative RT-PCR confirmed the presence of ORF1 mRNA in cultured RA SF (30-300-fold the amount in normal SF), demonstrating the existence of a nondegenerated and functional L1 element. In vitro, the majority of RA SF expressed ORF2/L1 mRNA. After incubation of SF with 5-azaC, L1 mRNA appeared in a time- and dose-dependent manner. Compared with OA SF, RA SF were more sensitive to 5-azaC. After transfection of RA SF with a functional L1.2 element, human stress-activated protein kinase 2 delta (SAPK2delta [or SAPK4]), met protooncogene, and ***galectin*** - ***3*** binding protein genes were differentially expressed. The transcription of the SAPK2delta gene, favored also by DNA hypomethylation in vitro, was confirmed in RA synovial tissues. CONCLUSION: Taken together, these data suggest that L1 elements and SAPK2delta pathways play a role in the activation of RA SF.

L3 ANSWER 18 OF 33 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 2001050304 MEDLINE
DOCUMENT NUMBER: 20517358 PubMed ID: 11062152
TITLE: Galectin-3 mediates genistein-induced G(2)/M arrest and inhibits apoptosis.

AUTHOR: Lin H M; Moon B K; Yu F; Kim H R
CORPORATE SOURCE: Department of Pathology and Breast Cancer Program, Barbara Ann Karmanos Cancer Institute, Wayne State University, School of Medicine, Detroit, MI 48201, USA.
CONTRACT NUMBER: CA64139 (NCI)
SOURCE: CARCINOGENESIS, (2000 Nov) 21 (11) 1941-5.
Journal code: 8008055. ISSN: 0143-3334.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001214

AB Many recent studies have focused on potential chemopreventive activities of dietary genistein, a natural isoflavonoid compound found in soy products. Genistein has been implicated in anticancer activities, including differentiation, apoptosis, inhibition of cell growth and inhibition of angiogenesis. In previous studies, genistein was shown to induce apoptosis and cell cycle arrest at G(2)/M in several cancer cell lines in vitro, which is associated with induction of p21(WAF1/CIP1), a universal ***inhibitor*** of cyclin-dependent kinases. At present, the molecular basis for diverse genistein-mediated cellular responses is largely unknown. In the present study, we investigated whether ***galectin*** - ***3*** , an anti-apoptotic gene product, regulates genistein-mediated cellular responses. We show that genistein effectively induces apoptosis without detectable cell cycle arrest in BT549, a human breast epithelial cell line which does not express ***galectin*** - ***3*** at a detectable level. In ***galectin*** - ***3*** transfected BT549 cells, genistein induced cell cycle arrest at the G(2)/M phase without apoptosis induction. Interestingly, genistein induces p21(WAF1/CIP1) expression in ***galectin*** - ***3*** -expressing BT549 cells, but not in control BT549 cells undergoing apoptosis. Collectively, the results of the present study suggest that ***galectin*** - ***3*** , at least in part, is a critical determinant for genistein-mediated cell cycle arrest and apoptosis, and genistein induction of p21(WAF1/CIP1) is associated with cell cycle arrest, but not required for apoptosis induction.

L3 ANSWER 19 OF 33 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:264406 BIOSIS
DOCUMENT NUMBER: PREV200000264406
TITLE: Inhibition of angiogenesis by a natural polysaccharide: MCP.
AUTHOR(S): Nangia-Makker, P. (1); Honjo, Y.; Hogan, V.; Raz, A.
CORPORATE SOURCE: (1) Karmanos Cancer Institute, Detroit, MI USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 2000) No. 41, pp. 645-646. print..
Meeting Info.: 91st Annual Meeting of the American Association for Cancer Research. San Francisco, California, USA April 01-05, 2000
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 20 OF 33 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 2000494114 MEDLINE
DOCUMENT NUMBER: 20342616 PubMed ID: 10878445
TITLE: Expression of galectin-3 in cells exposed to stress-roles of jun and NF-kappaB.
AUTHOR: Dumic J; Lauc G; Flogel M
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Faculty of Pharmacy and Biochemistry, University of Zagreb, Ante Kovavica 1, 10000 Zagreb, Croatia.
SOURCE: CELLULAR PHYSIOLOGY AND BIOCHEMISTRY, (2000) 10 (3) 149-58.
Journal code: 9113221. ISSN: 1015-8987.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001019

AB BACKGROUND/AIMS: ***Galectin*** - ***3*** is an interesting intracellular lectin that appears to be involved in numerous physiological processes. We have analyzed expression of ***galectin*** - ***3*** in glioblastoma cells exposed to heat-shock, alkylating agents, UV-C radiation and subculturing (trypsinization). METHODS: Protein levels of ***galectin*** - ***3*** were measured by western-blot analysis using M3/38 monoclonal antibody. The involvement of transcription factors NF-kappaB and Jun in the induction of ***galectin*** - ***3*** was addressed using specific ***inhibitor*** of NF-kappaB (zL(3)-vs) and antisense-jun oligonucleotides. RESULTS: Exposure of cells to heat-shock or subculturing (trypsinization) decreased levels of ***galectin*** - ***3*** to approximately 50%. Alkylating damage and UV-C irradiation caused an increase in the expression of ***galectin*** - ***3*** . Both inhibition of Jun by antisense-jun oligonucleotides, and inhibition of NF-kappaB by specific proteasomal ***inhibitor*** attenuated the induction of ***galectin*** - ***3*** by UV-light, but with somewhat different kinetics. CONCLUSIONS: We have found that different forms of cellular stress have different effects on the expression of ***galectin*** - ***3*** . Heat-shock and subculturing decrease, while alkylating agents and UV-light increase ***galectin*** ***3*** . NF-kappaB and Jun were shown to be involved in the induction of ***galectin*** - ***3*** by UV-light, which is a first demonstration that these transcriptional factors are involved in the regulation of ***galectin*** - ***3*** expression.

L3 ANSWER 21 OF 33 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 1999391262 MEDLINE
DOCUMENT NUMBER: 99391262 PubMed ID: 10463621
TITLE: Cell cycle arrest and inhibition of anoikis by galectin-3 in human breast epithelial cells.
AUTHOR: Kim H R; Lin H M; Biliran H; Raz A
CORPORATE SOURCE: Department of Pathology, Karmanos Cancer Institute, Wayne State University, School of Medicine, Detroit, Michigan 48201, USA.. hrckim@med.wayne.edu
CONTRACT NUMBER: CA46120 (NCI)
CA64139 (NCI)
SOURCE: CANCER RESEARCH, (1999 Aug 15) 59 (16) 4148-54.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19990925
Last Updated on STN: 19990925
Entered Medline: 19990916

AB ***Galectin*** - ***3*** is a member of a growing family of animal beta-galactoside-binding proteins shown to be involved in cell growth, differentiation, apoptosis resistance, and tumor progression. In the present study, we investigated whether ***galectin*** - ***3*** can protect against apoptosis induced by the loss of cell anchorage (anoikis). Because studies suggest that cellular sensitivity to anoikis is associated with cell cycle regulation, we examined the role of ***galectin*** - ***3*** on cell cycle regulation. Although BT549 cells (human breast epithelial cells) undergo anoikis, ***galectin*** - ***3*** -overexpressing BT549 cells respond to the loss of cell adhesion by inducing G1 arrest without detectable cell death. ***Galectin*** - ***3*** -mediated G1 arrest involves down-regulation of G1-S cyclin levels (cyclin E and cyclin A) and up-regulation of their inhibitory protein levels (p21(WAF1/CIP1) and p27KIP1). After the loss of cell anchorage, Rb protein becomes hypophosphorylated in ***galectin*** - ***3*** -overexpressing cells, as predicted from the flow cytometric analysis and immunoblot analysis of cyclins and their ***inhibitors*** . Interestingly, ***galectin*** - ***3*** induces cyclin D1 expression (an early G1 cyclin) and its associated kinase activity in the absence of cell anchorage. On the basis of these results, we propose that ***galectin*** - ***3*** inhibition of anoikis involves cell cycle

arrest at an anoikis-insensitive point (late G1) through modulation of gene expression and activities of cell cycle regulators. The present study suggests that ***galectin*** - ***3*** may be a critical determinant for anchorage-independent cell survival of disseminating cancer cells in the circulation during metastasis.

L3 ANSWER 22 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:550374 CAPLUS
DOCUMENT NUMBER: 132:93570
TITLE: Scope of multivalent ligand function. Lactose-bearing neoglycopolymers by ring-opening metathesis polymerization
AUTHOR(S): Pohl, Nicola L.; Kiessling, Laura L.
CORPORATE SOURCE: Dep. Chemistry, Univ. Wisconsin, Madison, WI, 53706, USA
SOURCE: Synthesis (1999), (Spec. Iss.), 1515-1519
CODEN: SYNTBF; ISSN: 0039-7881
PUBLISHER: Georg Thieme Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An understanding of both monovalent and multivalent carbohydrate-protein interactions is required for the design of effective ***inhibitors*** of protein-saccharide interactions. Here, a lactose-bearing norbornene imide template was polymd. using the Ru alkylidene catalyst, (Cy3P)2Cl2Ru:CHPh, to produce a lactose-substituted neoglycopolymer. The resulting polymer showed a 100-fold overall increase in inhibitory potency (5-fold increase on a per saccharide residue basis) compared to monomeric lactose in both a ***galectin*** ***3*** -binding assay and an Erythrina corallodendrum hemagglutination assay with its lactose-binding lectin.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 23 OF 33 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 2000005865 MEDLINE
DOCUMENT NUMBER: 20005865 PubMed ID: 10536041
TITLE: Lactose-containing starburst dendrimers: influence of dendrimer generation and binding-site orientation of receptors (plant/animal lectins and immunoglobulins) on binding properties.
AUTHOR: Andre S; Ortega P J; Perez M A; Roy R; Gabius H J
CORPORATE SOURCE: Institute of Physiological Chemistry, Faculty of Veterinary Medicine, Ludwig-Maximilians-University, Veterinarstrasse 13, D-80539 Munich, Germany.
SOURCE: GLYCOBIOLOGY, (1999 Nov) 9 (11) 1253-61.
Journal code: 9104124. ISSN: 0959-6658.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000131
Last Updated on STN: 20000131
Entered Medline: 20000119
AB Starburst glycodendrimers offer the potential to serve as high-affinity ligands for clinically relevant sugar receptors. In order to define areas of application, their binding behavior towards sugar receptors with differential binding-site orientation but identical monosaccharide specificity must be evaluated. Using poly(amidoamine) starburst dendrimers of five generations, which contain the p-isothiocyanato derivative of p-aminophenyl-beta-D-lactoside as ligand group, four different types of galactoside-binding proteins were chosen for this purpose, i.e., the (AB)(2)-toxic agglutinin from mistletoe, a human immunoglobulin G fraction, the homodimeric galectin-1 with its two binding sites at opposite ends of the jelly-roll-motif-harboring protein and monomeric ***galectin*** - ***3*** . Direct solid-phase assays with surface-immobilized glycodendrimers resulted in obvious affinity enhancements by progressive core branching for the plant agglutinin and less pronounced for the antibody and galectin-1. High density of binding of ***galectin*** - ***3*** with modest affinity increases only from the level of the 32-mer onwards points to favorable protein-protein interactions of the monomeric lectin and a spherical display of the end

groups without a major share of backfolding. When the inhibitory potency of these probes was evaluated as competitor of receptor binding to an immobilized neoglycoprotein or to asialofetuin, a marked selectivity was detected. The 32- and 64-mers were second to none as ***inhibitors*** for the plant agglutinin against both ligand-exposing matrices and for galectin-1 on the matrix with a heterogeneous array of interglycoside distances even on the per-sugar basis. In contrast, a neoglycoprotein with the same end group was superior in the case of the antibody and, less pronounced, monomeric ***galectin*** - ***3*** . Intimate details of topological binding-site presentation and the ligand display on different generations of core assembly are major operative factors which determine the potential of dendrimers for applications as lectin-targeting device, as attested by these observations.

L3 ANSWER 24 OF 33 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 1998225204 MEDLINE
DOCUMENT NUMBER: 98225204 PubMed ID: 9556610
TITLE: Galectin-1 is a major receptor for ganglioside GM1, a product of the growth-controlling activity of a cell surface ganglioside sialidase, on human neuroblastoma cells in culture.
AUTHOR: Kopitz J; von Reitzenstein C; Burchert M; Cantz M; Gabius H J
CORPORATE SOURCE: Institut fur Pathochemie und Neurochemie, Klinikum der Ruprecht-Karls-Universitat, Im Neuenheimer Feld 220, D-69120 Heidelberg, Germany.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 May 1) 273 (18) 11205-11.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980611
Last Updated on STN: 19980611
Entered Medline: 19980602
AB Cell density-dependent inhibition of growth and neural differentiation in the human neuroblastoma cell line SK-N-MC are associated with a ganglioside sialidase-mediated increase of GM1 and lactosylceramide at the cell surface. Because these glycolipids expose galactose residues, we have initiated the study of the potential role of galectins in such cellular events. Using specific antibodies, galectin-1 but not ***galectin*** - ***3*** was found to be present at the cell surface. Assessment of carbohydrate-dependent binding revealed a saturable amount of ligand sites approaching 2.6 x 10(6) galectin-1 molecules bound/cell. Presence during cell culture of the sialidase ***inhibitor*** 2-deoxy-2,3-dehydro-N-acetylneuraminic acid or of the GM1-binding cholera toxin B subunit effected a decrease of the presentation of galectin-1 ligands by 30-50%. The assumption that GM1 is a major ligand for galectin-1 was reinforced by the correlation between the number of carbohydrate-dependent 125I-iodinated GM1-neoganglioprotein binding sites and the amount of immunoreactive surface galectin-1, the marked sensitivity of probe binding to the presence of anti-galectin-1 antibody, and the inhibition of cell adhesion to surface-immobilized GM1 by the antibody. The results open the possibility that the carbohydrate-dependent interaction between ganglioside GM1 and galectin-1 may relay sialidase-dependent alterations in this cell system.

L3 ANSWER 25 OF 33 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:763758 SCISEARCH
THE GENUINE ARTICLE: 121HC
TITLE: Effects of protein kinase ***inhibitors*** and oncogenes on ***galectin*** - ***3*** expression in monocyte-macrophage cell lines.
AUTHOR: Kim K (Reprint); Mayer E P; Legrand A; Nachtigal M
CORPORATE SOURCE: UNIV S CAROLINA, SCH MED, COLUMBIA, SC 29208; UNIV ORLEANS, F-45067 ORLEANS, FRANCE
COUNTRY OF AUTHOR: USA; FRANCE
SOURCE: FASEB JOURNAL, (17 MAR 1998) Vol. 12, No. 4, Part 1, Supp. [S], pp. 2801-2801.
Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE

PIKE, BETHESDA, MD 20814-3998.
ISSN: 0892-6638.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L3 ANSWER 26 OF 33 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:201174 BIOSIS
DOCUMENT NUMBER: PREV199800201174
TITLE: Effects of protein kinase ***inhibitors*** and oncogenes on ***galectin*** - ***3*** expression in monocyte-macrophage cell lines.
AUTHOR(S): Kim, K. (1); Mayer, E. P.; Legrand, A.; Nachtigal, M.
CORPORATE SOURCE: (1) Univ. S. Carolina, Sch. Med., Columbia, SC 29208 USA
SOURCE: FASEB Journal, (March 17, 1998) Vol. 12, No. 4, pp. A482. Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 98, Part 1 San Francisco, California, USA April 18-22, 1998 Federation of American Societies for Experimental Biology . ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 27 OF 33 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 1998122543 MEDLINE
DOCUMENT NUMBER: 98122543 PubMed ID: 9462711
TITLE: Modulation of galectin-1 content in human head and neck squamous carcinoma cells by sodium butyrate.
AUTHOR: Gillenwater A; Xu X C; Estrov Y; Sacks P G; Lotan D; Lotan R
CORPORATE SOURCE: Department of Tumor Biology, The University of Texas M.D. Anderson Cancer Center, Houston 77030, USA.. agillenwater@hns.mdacc.tmc.edu
CONTRACT NUMBER: CA57166 (NCI)
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1998 Jan 19) 75 (2) 217-24. Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19990129
Entered Medline: 19980219
AB Galectin-1 and ***galectin*** - ***3*** are beta-galactoside-binding proteins thought to be important for cellular interactions, growth regulation and differentiation. Alterations in cellular content of galectins have been associated with differentiation, transformation and malignant progression. We examined the modulation of galectin-1 and ***galectin*** - ***3*** expression in head and neck squamous cell carcinoma (HNSCC) cell lines by treatment with sodium butyrate, a known differentiation-modulating agent, and identified potential mechanisms of butyrate regulation of galectin-1 levels in one of the cell lines. Sodium butyrate effected an increase in galectin-1 protein concentration in 5 of 8 cell lines. One cell line, MDA-886LN, showed a marked time- and dose-dependent increase from barely detectable amounts with butyrate treatment. Concurrently with increased galectin-1 expression, butyrate treatment promoted morphologic changes, induced growth inhibition and inhibited soft agar colony formation in MDA-886LN cells. Butyrate-treated MDA-886LN cells demonstrated increased galectin-1 mRNA content, suggesting a role for butyrate in transcriptional regulation of galectin-1 expression. Treatment with other ***inhibitors*** of histone deacetylase also induced an increase in galectin-1 expression. Together, our results indicate that butyrate treatment can modulate galectin-1 content in MDA-886LN HNSCC cells as well as induce morphologic changes and growth inhibition. This action may involve a combination of transcriptional regulation and inhibition of histone deacetylation.

L3 ANSWER 28 OF 33 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 1998111032 MEDLINE

DOCUMENT NUMBER: 98111032 PubMed ID: 9450571
TITLE: Preferential adhesion of prostate cancer cells to a human bone marrow endothelial cell line.
COMMENT: Comment in: J Natl Cancer Inst. 1998 Apr 1;90(7):547
Comment in: J Natl Cancer Inst. 1998 Jan 21;90(2):84-5
AUTHOR: Lehr J E; Pienta K J
CORPORATE SOURCE: University of Michigan Comprehensive Cancer Center, Department of Internal Medicine, Ann Arbor 48109-0946, USA.
CONTRACT NUMBER: CA60156 (NCI)
CA69568 (NCI)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1998 Jan 21) 90 (2) 118-23.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980224
Last Updated on STN: 19990129
Entered Medline: 19980210

AB BACKGROUND: In virtually all patients with advanced prostate cancer, the disease metastasizes to bone and causes osteoblastic growth. However, the mechanisms that contribute to bone metastasis are poorly understood. It has been hypothesized that the bone provides a favorable growth environment for prostate cancer cells, which nonselectively seed the bone marrow from the bloodstream. Alternatively, prostate cancer cells may preferentially bind to bone marrow endothelial cells. We developed an in vitro model of bone endothelium and tested the hypothesis that prostate cancer cells adhere preferentially to bone marrow endothelial cells. METHODS: We isolated and characterized a human bone marrow endothelial (HBME-1) cell line. Cells were transfected with the simian virus 40 large T antigen for immortalization. Cell surface receptors were characterized by immunohistochemistry and flow cytometry. The adhesion of cancer cells to HBME-1 and to endothelial cell lines from other organs was tested in an in vitro binding assay as were ***inhibitors*** of adhesion. RESULTS: The immortalized HBME-1 cell line demonstrated many properties characteristic of endothelial cells, including positive staining for von Willibrand factor and rapid formation of tubule structures when exposed to extracellular matrices. In an in vitro assay, prostate cancer cells adhered preferentially to human bone marrow endothelium when compared with endothelium derived from other sources. Preferential adhesion was blocked, in part, by antibodies to ***galectin*** - ***3*** and LFA-1. IMPLICATIONS: These data suggest that the propensity of prostate cancer cells to establish themselves in bone is due, at least in part, to their preferential adhesion to human bone marrow endothelial cells.

L3 ANSWER 29 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:775892 CAPLUS
DOCUMENT NUMBER: 128:113556
TITLE: Galectin-3: a novel antiapoptotic molecule with a functional BH1 (NWGR) domain of Bcl-2 family
AUTHOR(S): Akahani, Shiro; Nangia-Makker, Pratima; Inohara, Hidenori; Kim, Hyeong-Reh Choi; Raz, Avraham
CORPORATE SOURCE: Tumor Progression and Metastasis Program, Detroit, MI, 48201, USA
SOURCE: Cancer Research (1997), 57(23), 5272-5276
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Galectin-3, a .beta.-galactoside-binding protein, has been shown to be involved in tumor progression and metastasis. Here, the authors demonstrate that expression of galectin-3 in human breast carcinoma BT549 cells inhibits cis-diamminedichloroplatinum (cisplatin)-induced poly(ADP-ribose) polymerase degrdn. and apoptosis, without altering Bcl-2, Bcl-XL, or Bax expressions. Galectin-3 contains the NWGR amino acid sequence highly conserved in the BH1 domain of the bcl-2 gene family, and a substitution of glycine to alanine in this motif abrogated its antiapoptotic activity. The findings demonstrate that galectin-3 inhibits apoptosis through a cysteine protease pathway and highlight the functional significance of the NWGR motif in apoptosis resistance of a non-Bcl-2

protein.

L3 ANSWER 30 OF 33 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 1998073147 MEDLINE
DOCUMENT NUMBER: 98073147 PubMed ID: 9408830
TITLE: Further refinement of the description of the ligand-binding characteristics for the galactoside-binding mistletoe lectin, a plant agglutinin with immunomodulatory potency.
AUTHOR: Galanina O E; Kaltner H; Khraltsova L S; Bovin N V; Gabius H J
CORPORATE SOURCE: Shemyakin Institute of Bioorganic Chemistry, Russian Academy of Sciences, Moscow, Russian Federation.
SOURCE: JOURNAL OF MOLECULAR RECOGNITION, (1997 May-Jun) 10 (3) 139-47.
Journal code: 9004580. ISSN: 0952-3499.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980203

AB The galactoside-binding lectin from mistletoe (Viscum album L.) is a biological response modifier, eliciting e.g. enhanced secretion of cytokines. This immunological activity warrants the further analysis of its ligand-binding properties with special attention paid to blood group epitopes. To avoid the microheterogeneity and complexity of naturally occurring glycoproteins, chemically strictly defined neoglycoconjugates and a panel of synthetic oligosaccharides were employed in solid-phase assays for direct binding and assessment of the relative inhibitory capacity. Since label incorporation into the lectin, although performed under protective conditions, or surface immobilization by adsorption to plastic may affect its affinity characteristics, the extent of neoglycoconjugate binding in the absence of any interfering substance and in the presence of oligosaccharides was determined comparatively with labeled and with immobilized lectin. In principle, these two factors could be excluded to markedly alter binding features. In addition to lactose, the blood group determinants H and B were strongly reactive. A fucose residue can thus especially be accommodated to the binding site when linked to the non-reducing unit. N-Acetyllactosamine was nearly as potent as an ***inhibitor*** as lactose. Lec and the A determinant were notably inferior to the other ABH blood group epitopes. Le(a) and Le(x) and their sialylated derivatives displayed only very weak binding capacity. Among the two natural isomers of sialyllactose, the alpha 2,6-form displayed a higher level of inhibitory capacity than the alpha 2,3-derivative. Isomeric variants of the Thomsen-Friedenreich antigen, too, reduced lectin binding to the lactose-carrying polymer. Their capacities were surpassed by those of the H and the B determinants and a related form of the latter, the P1 epitope. An overlap of specificity with the immunomodulatory human ***galectin*** - ***3*** is thus measurable for H/B-like structures. The documented differential reactivity of the mistletoe lectin to blood group oligosaccharides may have a bearing on the responsiveness of blood group-positive cell populations.

L3 ANSWER 31 OF 33 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 97053673 MEDLINE
DOCUMENT NUMBER: 97053673 PubMed ID: 8898087
TITLE: Galectin-3 stimulates uptake of extracellular Ca2+ in human Jurkat T-cells.
AUTHOR: Dong S; Hughes R C
CORPORATE SOURCE: National Institute for Medical Research, London, UK.
SOURCE: FEBS LETTERS, (1996 Oct 21) 395 (2-3) 165-9.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961220

AB ***Galectin*** - ***3*** , a mammalian galactoside-binding protein, is not expressed in the Jurkat T-lymphoblastoid cell line. However, Jurkat cells express surface glycoprotein receptors for ***galectin*** - ***3*** , one of which is shown to be the glycosylated heavy chain of CD98 (4F2 antigen), a T-cell activation marker. Addition of ***galectin*** - ***3*** to Jurkat cells triggers a sustained influx of extracellular Ca2+ in a concentration dependent manner. The induced increase in cytosolic [Ca2+]i is blocked by sugar hapten ***inhibitors*** of ***galectin*** - ***3*** . The ***galectin*** - ***3*** -induced effect is insensitive to voltage-gated Ca2+ channel antagonists such as prenylamine, nifedipine and diltiazem and to pertussis toxin but is inhibited by cholera toxin. The results suggest that ***galectin*** - ***3*** released by accessory cells such as macrophages may bind in vivo to T-cell activation antigens and also participate in Ca2+ signalling.

L3 ANSWER 32 OF 33 MEDLINE DUPLICATE 18
ACCESSION NUMBER: 95156501 MEDLINE
DOCUMENT NUMBER: 95156501 PubMed ID: 7853416
TITLE: Inhibition of spontaneous metastasis in a rat prostate cancer model by oral administration of modified citrus pectin.
COMMENT: Comment in: J Natl Cancer Inst. 1995 MAr 1;87(5):331-2
AUTHOR: Pienta K J; Naik H; Akhtar A; Yamazaki K; Replogle T S; Lehr J; Donat T L; Tait L; Hogan V; Raz A
CORPORATE SOURCE: Division of Hematology and Oncology, Wayne State University School of Medicine, Detroit, Mich.
CONTRACT NUMBER: CA46120 (NCI)
CA57453 (NCI)
CA60156 (NCI)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1995 Mar 1) 87 (5) 348-53.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950322
Last Updated on STN: 19950322
Entered Medline: 19950316

AB BACKGROUND: Prostate cancer is the most common cancer diagnosed in U.S. men and remains incurable once it has metastasized. Many stages of the metastatic cascade involve cellular interactions mediated by cell surface components, such as carbohydrate-binding proteins, including galactoside-binding lectins (galectins). Modified citrus pectin (pH-modified), a soluble component of plant fiber derived from citrus fruit, has been shown to interfere with cell-cell interactions mediated by cell surface carbohydrate-binding ***galectin*** - ***3*** molecules. PURPOSE: The aim of this study was to determine whether modified citrus pectin, a complex polysaccharide rich in galactosyl residues, could inhibit spontaneous metastasis of prostate adenocarcinoma cells in the rat. METHODS: The ability of modified citrus pectin to inhibit the adhesion of Dunning rat prostate cancer MAT-LyLu cells to rat endothelial cells was measured by 51Cr-labeling. Modified citrus pectin inhibition of MAT-LyLu cell anchorage-independent growth was measured by colony formation in agarose. The presence of ***galectin*** - ***3*** in rat MAT-LyLu cells and human prostate carcinoma was demonstrated by immunoblotting and immunohistochemistry. One million MAT-LyLu cells were injected subcutaneously into the hind limb of male Copenhagen rats on day 0. Rats were given 0.0%, 0.01%, 0.1%, or 1.0% (wt/vol) modified citrus pectin continuously in their drinking water (from day 4 until necropsy on day 30). The number of MAT-LyLu tumor colonies in the lungs were counted. RESULTS: Compared with 15 or 16 control rats that had lung metastases on day 30, seven of 14 rats in the 0.1% and nine of 16 rats in the 1.0% modified citrus-pectin group had statistically significant (two-sided; $P < .03$ and $P < .001$, respectively) reductions in lung metastases. The lungs of the 1.0% modified citrus pectin-treated rats had significantly (two-sided; $P < .05$) fewer metastatic colonies than control groups (9 colonies +/- 4 [mean +/- SE] in the control group compared with 1 colony +/- 1 in the treated group). Modified citrus pectin had no effect on the growth of the primary tumors. In vitro, modified citrus pectin inhibited

MAT-LyLu cell adhesion to [redacted] endothelial cells in a time- and dose-dependent manner as well as their colony formation in [redacted] isolid medium. CONCLUSIONS: We present a novel therapy in which oral intake of modified citrus pectin acts as a potent ***inhibitor*** of spontaneous prostate carcinoma metastasis in the Copenhagen rat. IMPLICATIONS: Further investigations are warranted to determine the following: 1) the role of ***galectin*** - ***3*** in normal and cancerous prostate tissues and 2) the ability of modified citrus pectin to inhibit human prostate metastasis in nude mice.

L3 ANSWER 33 OF 33 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 94296411 MEDLINE
DOCUMENT NUMBER: 94296411 PubMed ID: 8024581
TITLE: Identification of human melanoma cellular and secreted ligands for galectin-3.
AUTHOR: Inohara H; Raz A
CORPORATE SOURCE: Cancer Metastasis Program, Michigan Cancer Foundation, Detroit 48201.
CONTRACT NUMBER: R01-CA46120 (NCI)
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1994 Jun 30) 201 (3) 1366-75.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940815
Last Updated on STN: 19970203
Entered Medline: 19940801

AB The endogenous human tumor-associated ***galectin*** - ***3*** (hL-31) is a functional molecule which acts as a receptor for ligands containing poly-N-acetyllactosamine sequences. However, little is known about its native ligand(s). In order to identify the ligand(s), the human melanoma cell line A375 was metabolically labeled with [3H]glucosamine, and total cell extracts and serum-free conditioned medium of the labeled cells were affinity-purified on immobilized recombinant hL-31 followed by elution with lactose, the specific sugar ***inhibitor*** of the lectin. Cellular ligands for hL-31 were found to be composed of the two lysosome-associated membrane proteins, LAMP-1 and LAMP-2, while secreted ligands consisted of two glycoproteins of 98 and 70 kDa. N-terminal protein microsequencing revealed that the 98 kDa and 70 kDa species share the same N-terminal sequence. The functional relevance of these secreted ligands was demonstrated by their ability to inhibit lectin-mediated hemagglutination in a manner similar to the specific sugar ***inhibitor*** lactose. Computer-assisted sequence library searches have identified the 98 kDa human melanoma secreted ligand to be the Mac-2-binding protein (Mac-2-BP), also known as the human lung tumor L3 antigen.

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(FILE 'HOME' ENTERED AT 10:23:54 ON 09 JUL 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 10:24:35 ON 09 JUL 2002
L1 1767 S GALECTIN-3
L2 93 S L1 (P) INHIBITOR
L3 33 DUPLICATE REMOVE L2 (60 DUPLICATES REMOVED)
L4 0 S L3 (P) COMPOSITION

=> log y

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